however, the spouses are not subject to the development of Crohn disease. Blood relatives, on the other hand, are.

I think this single observation is sufficient, by itself, to dispute an infectious etiology as the prime, that is initial, cause of Crohn disease even in the presence of other co-factors.

I would appreciate Dr. Cello's commentary.

ARNOLD L. FICK, MD
San Diego, California

* * *

Dr. Cello Replies

TO THE EDITOR: With respect to Dr. Flick's letter, the absence of a significantly increased incidence of Crohn disease in spouses of patients does *not* disprove absolutely an infectious etiology to Crohn disease. If unique transmissible agents are reproducibly isolated from patients with Crohn disease but not from controls (including cohabitor spouses) the importance of host susceptibility factors must definitely be taken into consideration.

Tissue culture and *in vivo* animal studies continue to isolate unique agents from patients with Crohn disease. These agents are not seen in control populations or in patients with other inflammatory or neoplastic bowel diseases. Moreover, these unique cytopathic and histopathic agents have been serially passaged more than ten times in experimental systems.[1] These study findings certainly need further confirmation with larger numbers of patients with Crohn disease before unequivocal statements can be made. A better definition of unique inherited factors may further clarify those risk factors necessary for developing clinical Crohn disease in persons exposed to these agents.

JOHN P. CELLO, MD
Chief, Clinical Gastroenterology
University of California Service
San Francisco General Hospital

REFERENCE

1. Gitnick GL: The course and character of viral infection in Crohn's disease and ulcerative colitis (abstr). Gastroenterology 74:1038, 1978

Editor's Note

DR. CELLO raised the point that in his original article Crohn's disease was changed to Crohn disease.

The WESTERN JOURNAL staff for the most part uses the *Stylebook/Editorial Manual of the AMA*, Sixth Edition,[1] as the basis for the journal's editorial style. The *Stylebook* specifically notes that "eponyms are not written in the possessive form" and give Wilms tumor, Graves disease, Hodgkin disease and Ewing sarcoma among other examples.

The sixth edition *Stylebook* was selected for several reasons. One reason is well stated in the book's foreword: "A scientific journal should have a consistency of style . . . on which readers come to rely. The few rules a journal adopts should be simple, inviolable, and encourage clear, unambiguous writing."

A second reason, and more specifically to the point of why this particular stylebook was chosen, is related to the observation that the English language is continually going through a process of simplification. When two usages or forms are possible, the simpler is preferable and, in fact, usually will win out in the end (arthro-empyesis and arthro-endoscopy in the 24th edition of *Dorland's Illustrated Medical Dictionary* [1965], arthroempyesis and arthroendoscopy in the 25th edition [1974]). The sixth edition AMA *Stylebook* recognizes this "simpler is preferable" school of journalism.

REFERENCE

1. Stylebook/Editorial Manual of the AMA, Sixth Edition—Prepared by the Scientific Publications Division, AMA, William R. Barclay, MD, Director. Publishing Sciences Group, Inc., 545 Great Road, Littleton, MA 01460, 1976 ($6.50)

Vitamin A and Cirrhosis

TO THE EDITOR: A title should accurately reflect the content of an article, as I have noted elsewhere.[1] The report "Cirrhosis Due to Hypervitaminosis A" (West J Med 128:244-246, Mar 1978) would more appropriately have been titled "Association of Cirrhosis and Vitamin A Ingestion." The authors do not prove a cause-and-effect relation between their patient's vitamin ingestion and his cirrhosis.

The clinical findings are suggestive of hypervitaminosis A but can have other causes. Dry skin, for instance, could have been related to the patient's biliary obstruction (with consequent fat malabsorption), and cheilosis can be caused by protein deficiency, which the patient exhibited. Chronic ingestion of 40,000 units of vitamin A by an adult would be unlikely to cause hypervitaminosis A; I believe there is only one other reported case[2] with that daily intake.

The hepatic changes are likewise not specific for vitamin A intoxication. Although Hruban and co-workers[3] observed sinusoidal fibrosis, perisinusoidal accumulation of Ito cells, atrophy of hepatocytes and other changes in the patients in their two cases, they point out that most of the changes have been observed in chronic diseases of the liver other than vitamin A intoxication.